After successive large pilocarpin doses there occurs a long continuing condition in which stimulation of the sympathetic causes no increase, but only a decrease of the secretion, more and more marked (in certain limits) with the increase in the strength of the stimuli and the time during which the stimuli are applied, and in which stimulation of the chorda causes a very slight increase in the secretion, rather more marked with the stronger stimuli. If a very large quantity of pilocarpin is brought into the gland either by direct injection into the gland (lingual) artery or into the gland duct, the secretion ceases at once, and the nerves are paralyzed for some time.

Atropin, in sufficient quantity, produces a complete stoppage of secretion and paralysis of the secretory nerves even after a previous dose of pilocarpin. The atropin effect can, however, be completely overcome by a subsequent injection of pilocarpin into the gland artery or duct. This mutual antagonism can be repeatedly shown, but is each time less perfect as far as the action of pilocarpin is concerned.

By stimulating either chorda or sympathetic (electrically) and causing a secretion, the atropin paralysis of these nerves, but especially of the sympathetic (which normally is less affected by atropin than the chorda), is more readily brought about. The atropin paralysis is also more readily effected after prolonged pilocarpin secretion, but after pilocarpin a somewhat larger dose of atropin is required to paralyze the chorda than is normally the case. There are two factors then to be considered in the paralysis: the amount of pilocarpin—this causes the atropin to produce its effects less readily,—and the amount of saliva secreted, which enables the atropin to produce its effects more readily.

The diminution of sympathetic effect produced by pilocarpin is unaffected by a small dose of atropin, unless some amount of secretion has taken place, but in the latter case the parts which the atropin attacks, be they nerve endings or gland cells, are more readily attacked by atropin, and an atropin paralysis adds itself to the pilocarpin diminution of sympathetic effect. But although pilocarpin in comparatively small quantity, when producing a secretion tends to paralyze the sympathetic, yet it but slightly affects it if a small amount of atropin (not enough by itself to paralyze that nerve) be given previously or at the same time.

As general conclusions, Langley adds:

Pilocarpin, in proportion to the quantity given, paralyzes both the chorda and sympathetic secretory fibres.

The diminution of the pilocarpin secretion caused by stimulating the sympathetic, is a direct effect of diminished blood supply, and not of nerve fibres inhibitory to the secretion.

The slight increase of saliva obtained by stimulating the chorda after a large dose of pilocarpin, is due, not to the action of its secretory, but of its vaso-dilator fibres. H. G.

NERVE DEGENERATION.—Notwithstanding the large number of researches on this subject, Dr. Th. Rumpf (*Unters. aus d. Phys. Institut d. Univ. Heidelberg*, II., 3, p. 307) undertook an investigation on the changes in divided

nerves, as a sequel to his researches on normal nerves (this journal, Jan., 1879).

Twenty-four hours after division of the sciatic nerve of the frog, the peripheral stump shows an escape of myeline, involving the fibres usually up to the first annular constriction, though sometimes also further. The following interannular segment (or at least a part of it), is filled with granules and cannot be colored with osmic acid. The axis cylinder is not visible until after the removal of the myeline by means of alcohol and ether. It is then found considerably swollen, and terminating with a club-shaped end a short distance from the point of division. The swelling of the axis cylinder extends through one or more interannular segments. As Rumpf had shown previously, the axis cylinder, after separation from the centre, is tumefied and finally dissolved by lymph, hence the thickened club-shaped end. In this gradual swelling Rumpf seeks the motive force for the escape of myeline.

Forty-eight and seventy-two hours after the section the myeline has diminished in quantity, and is but feebly colored by osmic acid. The absorption of myeline is partly the work of migrating cells, which enter the nerve fibre and transport fragments of myeline. By this time more of the axis cylinder has also been absorbed, while the swelling extends further along its course. The same observations can be made on the central stump of the nerve.

The degenerative changes proper occurring in the peripheral trunk of the divided nerve are of long duration and slow course in the frog. Even seven to eight days after the division the axis cylinder is not yet absorbed wholly in the interannular segment involved in the section.

An entirely different course is observed if the nerve be divided at two places, *i. e.*, the portion observed be cut off from both centre and periphery.

While the metamorphoses of the myeline are as usual, the axis cylinder swells rapidly and is completely absorbed in the course of a few days after a double division of the nerve. Hence, Rumpf concludes that the axis cylinder by itself has not the ability to regulate its own nutrition, but that its nutritive regulation depends on its connection with the centre. After separation from the centre, a certain but insufficient nutritive influence is still exerted by the peripheral termination.

Ranvier has claimed that the loss of conductibility of a divided nerve is due to the interruption of continuity of the axis cylinder by the encroaching protoplasm (this journal, Jan., 1879). Rumpf, however, rejects this view, since a divided nerve loses its irritability in the frog within fourteen days, while no interruption of continuity of the axis cylinder can be demonstrated even after sixteen days. Hence, while the nerve is unable to conduct motor impulses, a nutritive impulse from the periphery can still traverse it.

The horn sheaths of the nerve persist for quite a time even after complete solution of the axis cylinder. Finally, however, they disintegrate into granules or plaques, and disappear.

In mammals degenerative changes pursue a faster course than in the frog. Still the axis cylinder is but little changed the second day after a simple

division. Two days, however, after a double section, the axis cylinders are part swollen by the action of the lymph, and part completely dissolved.

THE COURSE OF SWEAT NERVES AND VASO-MOTOR FIBRES.—Induced by various contradictory statements, B. Luchsinger (*Pflüger's Arch.*, Vol. 18, XXXI., p. 483) has once more examined the course of the sweat nerves in the cat. He had previously claimed that the sweat nerves of the extremities were entirely derived from the sympathetic. This was confirmed by Ostroumoff and by Nawrocki. Adamkiewicz, however, and Vulpian had also found sweat nerves in the spinal roots of the extremity-nerves.

To decide this question a larger number of cats were examined. The abdominal sympathetic of one side was divided, and the animal either allowed to recover during some weeks or immediately tested, by placing it in a heated box. In the large majority the paw innervated by the divided sympathetic did not perspire in the least.

But in about one case in every six some sweat fibres did actually enter the nerves (of fore and hind extremity) through some route other than the sympathetic. In a second article, Luchsinger and Puelma (*Ibid*, p. 489) followed the course of the vaso-motor fibres of the sciatic nerve of the cat. As an unexceptional result they found that the nerves going to the vessels of the hind extremity are derived both from the sympathetic and the spinal roots.

DIRECT IRRITATION OF NERVE CENTRES has been studied by R. Marchand (*Pflüger's Archiv*, Vol. 18, XII., p. 511). In the first place, the frog's ventricle, separated from the greater part of the auricles, but still containing the ganglia situated at the auriculo-ventricular border, was compared with the ventricular muscle without ganglia. Mechanical, chemical, thermic or faradic irritation will induce but a single contraction in the heart muscle free from ganglionic cells, as it is obtained by isolating the lower part of the ventricle. If, however, the preparation still contains the ganglia, these stimuli cause a series of contractions. In the case of electric stimulation (a single induction shock) the number of pulsations increased directly with the degree of stimulation.

As a second test object, the spinal cord of decapitated frogs was employed. While a single induction shock applied to a motor nerve produces but a single muscle contraction, the same stimulus applied to the spinal cord provokes an irregular tetanus of some duration.

THE ACCELERATOR NERVES OF THE HEART.—Stricker and Wagner, *Mediz. Jahrb.*, Hft. 3, 1878 (abstr. in *Revue des Sci Méd.*).

It is now known that, in the dog, the acceleration of the pulse is produced by excitation of the *anse de Vieussens*, a nervous branch connecting the last cervical ganglion of the sympathetic to the first thoracic ganglion, or stellate ganglion. In order to find the real origin of the accelerator fibres of this branch, the authors isolated in the chest of the dog the trunk of the great